PHILADELPHIA NEUROLOGICAL SOCIETY.

December 20th, 1897.

President, Dr. Charles W. Burr, in the chair.

Dr. James Hendrie Lloyd presented a case of

ASTASIA-ABASIA.

The patient, a female, had much difficulty in standing and walking. Under suggestive therapeutics her condition had improved.

DISCUSSION.

Dr. Wharton Sinkler thought that the gait and whole attitude of this patient were very characteristic of the hysterical affections of gait. He stated that these vary greatly in degree and character, but this case he regarded as a type of the so-called astasia-abasia. In hysterical disturbances of gait there is usually a tendency to pitch to one side or backward, but the patient seldom falls or completely loses her balance, unless there is some one near to catch her.

Dr. David Edsall, by invitation, read a paper on

DISSOCIATION OF SENSATION, OF THE SYRINGOMYELIC TYPE, IN POTT'S DISEASE.

DISCUSSION.

Dr. Spiller stated that a few years ago it was thought that syringomyelia could be positively diagnosticated by dissociation of sensation; but, as Dr. Edsall's case and many others show, the difficulty in making this diagnosis increases. This dissociation of sensation occurs in tumors of the cord, in tabes, in disseminated sclerosis, in hysteria, in neuritis, and has been observed in the peculiar disease of the spinal column reported by v. Bechterew and Strümpell.

He did not think that the diagnosis of a tumor in this case could be made with any certainty. A spinal growth might be present and cause few symptoms, inasmuch as a tumor of the oblongata may be latent for some time. It seemed to him that all the symptoms could be produced by a lesion of the posterior roots, or by compression of the cord. This suggested the interesting theory of Goldscheider in regard to sensation, according to which pain is simply a summation of tactile impulses. Where pain sense is lost, and tactile sense is present, as in tabes, the posterior roots, though degenerated, permit the transmission of certain impulses, but not of a sufficient number to cause the perception of pain. This theory has, by no means, been accepted by all neurologists.

Dr. Hinsdale remarked that, about three years ago, Dr. Lloyd had shown, at one of the meetings of the Society, two cases of traumatic lesions of the spinal cord with syringomyelic symptoms. He thought that it is quite possible that in such cases there is extravasation of blood—a hæmatomyelia, which, after a time, is absorbed, but gives temporarily the dissociation symptom. This symptom does not persist, but causes a good deal of difficulty in forming a diagnosis while it lasts.

Dr. Eshner said that the fact that the sensory symptoms receded appeared to be against the probability of the existence of a tumor within the cord. One would not expect even a tuberculoma to diminish in size or to disappear within such a short time.

Dr. Burr spoke of the frequency of new growths, as shown by the necropsy, which have not been diagnosticated during life.

Dr. Edsall thought that it was quite possible that a tumor might have formed since the caries developed. In some tuberculous tumors that have been reported the growth has been very rapid. The peculiar distribution of the hypalgesia and thermal anæsthesia, and their extension to lower parts of the body, had led him to suspect the presence of a new growth. These could be better explained by the presence of a tumor than by any other means, provided the tumor originated in the centre of the cord and grew outwards.

Dr. W. E. Hughes, by invitation, reported

TWO CASES OF TUMOR OF THE BASE OF THE BRAIN,

and exhibited one of the specimens. A growth, the size of a hen's egg, was situated in the pituitary body. No symptoms of akromegaly had been observed during life.

DISCUSSION.

Dr. Hare desired to know how much of the pituitary body was left intact. If it is true that akromegaly often arises from disease of the pituitary body, it may be that when a certain portion of this body is left intact the symptoms do not develop. In the same way, if the pancreas is destroyed diabetes may appear, while if a portion is left pancreatic diabetes may not develop.

Dr. Hughes was unable to say how much of the pituitary body was involved.

Dr. A. A. Eshner made

A FURTHER CONTRIBUTION UPON A CASE OF MENINGITIS.

The report of this case was published in the Journal of Nervous and Mental Disease for March, 1897, p. 167. The patient died in convulsions, and a necropsy was made by Dr. Cattell.

On removing the calvarium the dura mater was not found adherent to the bone in this situation. The vessels generally were greatly injected, and the capillaries were somewhat more prominent than normal. All of the sulci in the Rolandic area on both sides exhibited a grayish-white, delicate, fibrous reticulum, which was slightly elevated on account of the presence of subjacent fluid. There was no indication of any process suggestive of recent bacteriologic involvement. The appearances were rather those of chronic fibrous thickening of the pia-arachnoid, with moderate adhesion to the brain surface. The blood vessels forming the circle of Willis were thickened, and gaped when cut. Many of them contained small fibrous nodes. The fibrous thickening of the meninges previously referred to was especially pronounced in the neighborhood of the basilar artery and upon the pons and medulla, the nerves coming off from which passed through the thickened membrane.

The under surface of the cerebrum, the olfactory, and the temporal regions were remarkably free from thickened membrane, which was, however, prominent in the fissure of Sylvius along the course of the middle cerebral artery. About three-quarters of an inch from the point of origin of the left middle cerebral artery was a small, well-formed sacculated aneurism, about the size of a small pea. The island of Reil and the retroinsular convolutions displayed no abnormality, other than atheroma of the blood vessels, and a small clot of blood on the right side, apparently contained within a miliary aneurism. There was considerable œdema in the neighborhood of the optic chiasm, and the optic nerve appeared flattened from above downward and diminished in size, from thickening of the overlying adventitious membrane.

The left lobe of the cerebellum was the seat of an enormous hemorrhage, which had ploughed its way into the fourth ventricle, and thence through the iter into the third and also into the left lateral ventricle. So enormous was this extravasation that a considerable clot of blood was found in the posterior horn of the left ventricle. The foramen of Munro and the adjacent nervous tissues were torn apart and replaced by a bloodclot. Sections through all parts of the brain failed to disclose any other seat of hemorrhage, recent or remote. The injection of some portions of the arbor vitæ of the cerebellum was especially pronounced.

Concerning the other organs, it seems only worth saying that the kidneys exhibited an extremely slight degree of parenchymatous inflammation, while the heart was somewhat enlarged, with healthy valves and orifices, and the lungs were œdematous and emphysematous.

Owing to the conditions under which the autopsy was held, it was not possible to obtain even the smallest portion of tissue for more careful study. It could not be decided from the macroscopic appearances whether or not the upper portion of the spinal cord exhibited any changes, particularly in its posterior columns.

Dr. E. Schreiner presented

A CASE OF ARSENICAL NEURITIS FOLLOWING THE TREATMENT OF CHOREA WITH FOWLER'S SOLUTION.

The patient, a female of ten years af age, was seen April 17th, 1896, in her second attack of chorea. She was placed on Fowler's solution, beginning with five drops three times daily. This was increased to twelve drops, when symptoms of the physiological action were observed, and the quantity was reduced. The total amount taken was two or three ounces. She remained under occasional observation until June 5th, when she showed loss of power in the arms and in the legs, more marked in the latter, with areas of pigmentation over the joints, some atrophy of the leg muscles, loss of patellar and plantar reflexes, diminution of tactile sensation, and marked reaction of degeneration, viz., complete loss of irritability to the faradic, and very slight response to strong galvanic currents.

There was a history of pain in the calves of the legs and of swelling below the knees two weeks before the patient's admission to the hospital. The only sensory symptom present on admission was some soreness in the course of the radial nerves.

Motor power and sensation improved, the latter more rapidly than the former, so that the patient finally was able to walk, though she presented the steppage gait. At present, December, 11th, 1897, the electrical reactions in the muscles of the legs and forearms are absent, and the muscular power, especially in the extensors of the leg, is imperfect, causing foot drop and steppage gait, but sensation is normal.

DISCUSSION.

Dr. Charles K. Mills said that this case interested him, especially in connection with the second question referred to, that is, as to the nature of the pathological changes which are probably present, and as to the names which should be used in speaking of cases of this kind. He thought that the time had come for us to revise our nomenclature of some of these toxic and infectious diseases of the nervous system. We certainly have very different types of cases which, without much thought, we class under the same names. Formerly, more than at present, these cases were spoken of as instances of myelitis; now we are more inclined to speak of them as cases of peripheral neuritis. It is to this point that he wished to ask attention.

He referred to a man who several years ago presented signs of arsenical poisoning. He got well after a time and left the hospital. Recently he came back with conditions similar to those referred to to-night. He had been addicted to the use of alcohol, and had been working as a painter, largely in white lead, for six or seven years. He had double foot drop, lost knee jerks, some irregularly disseminated disturbances of sensation, particularly dissociated anæsthesia, slight paresis of the upper extremities, and a little circumscribed pain in the limbs. Dr. Mills stated that a short time ago this case would have been looked upon by some as one of multiple neuritis; earlier, as one of myelitis; or, perhaps, it might have been regarded as a case which showed a concurrence of neuritis and myelitis.

The point which he wished to emphasize is that we need a new designation for these cases. We need to reform our nomenclature and classification of cases in which, as the result of an infection, or a toxæmia, or the direct action of a metallic poison, we have symptoms such as were reported tonight. The only sensory evidences of true neuritis in this case were the pain and hyperæsthesia, which, however, were not marked and were circumscribed; although the paralysis and loss of electrical reaction might also be regarded as due to neuritis.

In naming these diseases the idea should be enforced that the toxine, infection, or poisonous substance has set up a process which has led to the destruction of the peripheral neuron. Something circulating in the blood of the patient attacks the nerve cells—both cell body and processes, poisoning and destroying them, but not necessarily giving rise to inflammation. It is difficult to have clear ideas with regard to myelitis and neuritis on account of the different uses of terms.

Dr. Mills said that if he were asked what he would call cases of this kind, it would be a little difficult to say. We know that the chief evidences of inflammation are found in such conditions as hyperæmia, swelling and round cell infiltration. If these were present, we could properly class the disease as inflammatory. In their absence it is doubtful whether they should, or should not, be regarded as inflammatory affections. In reported cases both the peripheral nerves and the ganglion cells in the anterior horns of the cord have been found destroyed, or degenerated, without the usually accepted evidences of inflammation. It is, therefore, an open question whether these cases should be designated as neuritis, myelitis, or myleoneuritis; or whether, indeed, it would not be better to invent an entirely new term for them. Provisionally, he suggested that they should be called cases of *periphero-neuronal degeneration*, with the prefix of *acute*, or *chronic*, according to the intensity and rapidity of the destructive process.

Dr. Wharton Sinkler thought that Dr. Mills was going backward if he advocated the theory of myelitis in cases of toxic poisoning. It certainly is impossible to explain many of the curious symptoms present by regarding the lesion as located in the cord. It seemed to him that in a case like the one reported by Dr. Schreiner the view of peripheral neuritis is sufficient to account for the symptoms. In myelitis there is not apt to be as great loss of electrical irritability, nor is there the peripheral pain, hyperæsthesia and foot drop.

Dr. A. E. Taylor alluded to the recent address of Virchow upon inflammation, and, commenting upon our ignorance of many aspects of that process, expressed doubt as to whether the absence of round-celled infiltration could be relied upon as a criterion of the non-inflammatory nature of a pathological condition. The relations of inflammation to the interstitial and parenchymatous tissues would be clear if we knew exactly what inflammation always is, and what the parenchyma and interstitial tissues are.

Dr. Spiller stated that in many cases we are at present utterly unable to say whether a poison exerts its influence, primarily, on the nerve fibre or the cell from which this arises. He referred to the selective power of certain poisons for certain nerves, as seen in saturnine, alcoholic, diphtheritic and other forms of poisoning. He stated that probably all reported cases of recovery from tabes, such as are seen after diphtheria, are examples of pseudo-tabes, due to inflammation of sensory nerve fibres, and that certain poisons seem thus to pick out one set of fibres, even within a nerve, to the exclusion of others. He referred to the many experiments which have shown that, when a motor nerve is cut, the cell with which it is connected degenerates very rapidly. This degeneration may occur within a few hours after section of the motor nerve, and there is no reason for believing that these cellular changes are of an inflammatory character. They are changes in the chromophilic elements. He said that he had recently studied a case of compression of the cord, high up in the cervico-thoracic region, in which the direct cerebellar tracts were degenerated below the lesion, and the cells of the columns of Clarke had entirely disappeared. This, however, was in the nature of a retrograde degeneration in sensory fibres. He spoke of a case of acute neuritis, which he was then studying, and stated that the changes in the nerves were very great, and that the cells within the spinal cord presented chromatolysis.

Dr. Hare said that the fact that different poisons attack different parts of the nervous system is one which hardly admits of debate, and also that we have a pretty well-grounded idea that these poisons produce a primary neuritis, or some similar change. Many of the changes which occur afterward, and appear to be centred in the higher nervous system, are, perhaps, also due to the poison, or, perhaps, due to what may be called an ascending degeneration, a degeneration secondary to the peripheral degeneration.

He wished to know from Dr. Schreiner whether he noticed that the pigmentation in his case had any relation to the distribution of the anæsthesia, and whether these areas of pigmentation were peculiar in localization. It was taught at one time that the areas of pigmentation and anæsthesia are in relation to the joints. In arsenical neuritis, for example, it has been said that the anæsthesia in the upper extremity has a tendency to stop at the distal joint; if it goes beyond this, it stops at the elbow.

Dr. Mills said that he did not believe that the points which he had made had been met. He, of course, admitted that in many cases there is widespread peripheral involvement. In a case such as Dr. Spiller mentioned, where we find the peripheral nerves degenerated, and the cells in the anterior horns just as seriously degenerated, to his mind nothing justified us in calling these cases either neuritis or myelitis. If the law is true that pathological processes tend to follow in the line of physiological action, it is more probable that the toxic influence would be exerted from the centre toward the periphery in a motor nerve than the reverse. In any case, it must be acknowledged that the entire neuron is usually implicated. He desired to know from Dr. Spiller the intensity of the changes in the case he had referred to.

Dr. Spiller replied that he had examined the peripheral nerves, but was unable to say from "teased" preparations whether the fibres were inflamed or degenerated. He thought that it is very difficult to distinguish between the two conditions as regards the changes found in the nerves. He had noticed that the axis cylinders were destroyed, that some of the sheaths were swollen and filled with an increased amount of protoplasm and degenerated myelin, or else contracted and empty, and that the medullary substance was much enlarged. His examination had not yet revealed engorgement of vessels and round-cell infiltration. He said that the chromophilic elements in the nerve cells from which these fibres arose were broken up, and that the nucleus was displaced. He was unable to decide whether the cell was primarily or secondarily affected, inasmuch as similar lesions have been noted in both primary and secondary degeneration.

Dr. Schreiner stated that his reason for describing this as a case of neuritis was the fact that in recent literature cases of arsenical paralysis are described as neuritis, and because in three cases, the pathology of which he was able to look up, degenerative changes were found in the spinal nerves.

He stated in reply to the question of Dr. Hare that the pigmentation in the axilla was confined by the anterior and posterior borders of the axilla. At the elbow it formed a rather irregularly oval-shaped patch, extending above and below the crease of the elbow joint. There were no areas of pigmentation at the wrist joint. The pigmentation was limited by the boundaries of the popliteal space, and extended irregularly over the ankle.